

## Case report

# Tetany in association with gentamicin therapy

O L Beatty, N P S Campbell, R D G Neely

Accepted 1 March 1989.

---

Magnesium plays a role in calcium metabolism and hypomagnesaemia can lead to hypocalcaemia.<sup>1</sup> Recently it has been recognised that prolonged aminoglycoside therapy can cause hypomagnesaemia.<sup>2, 3</sup> We have recently observed tetany associated with low serum levels of both calcium and magnesium in a patient receiving gentamicin, which resolved when the gentamicin was stopped and the serum magnesium was corrected.

## CASE HISTORY

A 21-year-old male with a congenital ventricular septal defect was admitted with a ten-day history of rigors, malaise and anorexia. Three out of six blood cultures grew *Staphylococcus aureus*. A diagnosis of infective endocarditis was made and treatment with intravenous flucloxacillin and gentamicin was begun. Frequent peak and trough gentamicin levels were maintained within the therapeutic range. Eighteen days after treatment commenced, the pyrexia recurred and flucloxacillin was replaced by vancomycin. Three days later, he developed an allergic drug rash, and the vancomycin was changed to fusidic acid. Antibiotics were administered for a total of 56 days (Table).

Fifty-one days after treatment commenced, he complained of tingling around the lips and developed carpopedal spasm. The tetany responded to 30ml 10% calcium gluconate (6.69mmol). The uncorrected serum calcium was 1.36mmol/l (2.10–2.50) and the serum magnesium was 0.39mmol/l (0.7–1.03). Serum parathormone was 0.22µg/l which is inappropriately low for the serum calcium. Serum urea was 7.7mmol/l (3.3–8.8), creatinine 154mmol/l (40–110), and albumin 39g/l (30–45).

A magnesium load of 30mmol/l magnesium sulphate in 250ml of 5% dextrose was given intravenously over four hours, and urine collected over 24 hours for magnesium excretion. He excreted 68% of the total magnesium load in 24 hours (normal less than 50%) which shows excessive renal magnesium loss and excludes non-renal causes of magnesium deficiency.<sup>1, 4</sup> The calculation of the magnesium deficit is empirical as 99% of total body magnesium is intracellular and so serum magnesium does not accurately reflect total body magnesium. The deficit required to produce hypomagnesaemia is 0.5–1 mmol/kg of body weight. In our patient the deficit was 50mmol.

---

Regional Medical Cardiology Centre, Royal Victoria Hospital, Belfast, BT12 6BA.

Olivia L Beatty, MB, Senior House Officer.

N P S Campbell, MD, MRCP, Consultant Cardiologist.

Department of Biochemistry, Royal Victoria Hospital, Belfast, BT12 6BA.

R D G Neely, BSc, MB, MRCP, Registrar.

Correspondence to Dr Beatty.

TABLE

*Gentamicin and other drug therapies in relation to serum calcium and magnesium*

<i>Day</i>	<i>Drugs</i>	<i>Serum Ca mmol/l</i>	<i>Serum Mg mmol/l</i>
1	Gentamicin, flucloxacillin	2.11	0.84
18	Gentamicin, vancomycin	2.19	0.89
21	Drug rash	2.11	0.71
28	Gentamicin, fusidic acid	2.12	0.74
35	Gentamicin, fusidic acid	2.11	0.79
46	Gentamicin, fusidic acid	1.96	0.62
48	Gentamicin, fusidic acid	1.61	0.66
51	Gentamicin, fusidic acid (onset of tetany, gentamicin stopped)	1.36	0.45
52	Magnesium sulphate infusion	1.63	0.98
55	Magnesium sulphate infusion	1.62	0.86
56	Magnesium glycerophosphate orally	1.71	0.87
80	Magnesium glycerophosphate orally	2.29	0.79

However, to replace this deficit, excess magnesium had to be administered as our patient only retained 32% of a magnesium load. Therefore he required at least 150 mmol magnesium in order to retain 50 mmol magnesium. A total of 210 mmol magnesium sulphate was given intravenously over five days. No further tetany occurred and calcium supplements were not required. He was then given oral magnesium glycerophosphate for three months.

## DISCUSSION

Magnesium deficiency can be the result of decreased intake (prolonged intravenous feeding, chronic alcoholism, intestinal malabsorption), increased non-renal loss (chronic diarrhoea, biliary fistula) or increased renal loss (diuretic therapy, renal tubular acidosis, drug-induced tubular injury [cisplatin, amphotericin, gentamicin], hyperaldosteronism).

In our patient, the evidence pointed to increased renal loss as a cause of magnesium deficiency as there was excessive renal excretion of magnesium, despite a low serum magnesium. The normal response of the kidney to hypomagnesaemia is to reabsorb the ion in the renal tubules. He had been on a diuretic from admission but this was later discontinued after a drug rash developed. Magnesium levels during administration of the diuretic were within the normal range. A small number of case reports show that prolonged aminoglycoside therapy is thought to cause a renal tubular leak of magnesium assessed by the renal response to a magnesium load.<sup>2, 3, 5</sup> Renal function may otherwise be entirely normal and toxic levels of the drug do not have to occur.

Hypomagnesaemia can cause hypocalcaemia by inhibiting the secretion of parathormone and by causing skeletal and renal resistance to the effects of parathormone.<sup>1</sup> Tetany in these patients will respond to intravenous magnesium sulphate and may not respond to intravenous calcium.<sup>2</sup> In our patient there was

an initial temporary response to intravenous calcium, but his hypocalcaemia was finally corrected by replacing magnesium, and oral calcium supplements were not required. It is suggested that all patients on prolonged aminoglycoside therapy should have regular measurements of serum calcium and magnesium. The renal tubular leak can persist up to three months and therefore prolonged oral magnesium therapy may be required.

#### REFERENCES

1. Berkelhammer C, Bear R. A clinical approach to common electrolyte problems: hypomagnesaemia. *Can Med Assoc J* 1985; **132**: 360-8.
  2. Wilkinson R, Lucas G, Heath D, Franklin I, Boughton B. Hypomagnesaemia tetany associated with prolonged treatment with aminoglycoside. *Br Med J* 1986; **292**: 818-9.
  3. Watson A, Coffey L, Keogh B, McCann S. Severe hypomagnesaemia and hypocalcaemia following gentamicin therapy. *Ir Med J* 1983; **79**: 381-3.
  4. Bar RS, Wilson HE, Mazzaberi EL. Hypomagnesaemic hypocalcaemia secondary to renal magnesium wasting. *Ann Intern Med* 1975; **82**: 646-9.
  5. Patel R, Savage A. Symptomatic hypomagnesaemia associated with gentamicin therapy. *Nephron* 1979; **23**: 50-2.
- 

#### BOOK REVIEW

**Insurance handbook for the medical office.** By Marilyn Takahashi Fordney. 3rd ed. (pp 528. Illus, figs. £18.00, paperback). Philadelphia: Saunders, 1989.

I approached this book with interest because American-type medical insurance practices are rapidly approaching our shores. My curiosity was even further whetted on reading through the table of contents when I found Chapters 2 and 3 related to *Coding for professional services* and *Diagnostic coding* and Chapter 4 dealing with *Diagnosis related groups* (DRGs). In these there is an excellent 'Idiot's guide' to the coding systems ICD-9 and ICD-9-CM, and a very full and general description of the rationale and working of DRGs.

These three chapters are, however, only 65 pages in a book of a total of 515, the remainder of which comprise a detailed and comprehensive exposition and explanation for the medical assistant, account's clerk and assistant treasurer in the North American medical domain. For them I am sure this is an update, easy to read and understand, which will enhance the skills necessary to handle medical insurance claims by physicians, surgeons and other doctors and their supporting staffs. It certainly will live up to its claim to help increase accuracy and efficiency on the job and thereby bring more revenue into the medical office. Nevertheless, interesting as it may be, it is not yet a required or recommended text for the British or Irish reader.

GAM